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Zearalenone occurrence and human exposure

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Abstract

Among the mycotoxins zearalenone (ZEA) is of interest because of the oestrogenic effects that it, and certain of its metabolites possess. The fungi that produce ZEA are found worldwide, particularly in cereal grains and derived products. This has prompted many surveys to detect these compounds in commodities and foods. As a result, the widespread occurrence of ZEA in foods is well documented. Previous summaries including extensive reports by the Joint FAO/WHO Expert Committee on Food Additives (JECFA), the European Commission's Scientific Cooperation on Questions Relating to Food (SCOOP), and others, have provided significant information on the occurrence of ZEA in commodities and foods. Publication of occurrence data has continued at a rapid pace, and certain of that data, as well as highlights from previous intake and exposure assessments, are summarised herein. Comparing estimates of intake (exposure) with previous estimates of tolerable daily intakes, suggests that, for many of the countries where exposure assessments have been done, the populations are exposed to levels that would be considered safe. The situation may be different in populations that consume large quantities of foods that are susceptible to contamination, or in instances where contamination is atypically high. For much of the world estimates of exposure have not been reported, meaning that for much of the world, the true extent of the relevance of ZEA to human health remains uncharacterised.

Keywords: zearalenone, zearalenol, tolerable daily intake, human health, exposure assessments

1. Introduction

Zearalenone (ZEA) is a non-steroidal oestrogen produced by many species of *Fusarium* fungi that commonly infest cereal grains, in particular *F. graminearum, F. culmorum, F. equiseti and F. crookwellense* (Hagler *et al.*, 2001). Infestation is especially prevalent in temperate climates when relatively cool temperatures and high humidity coincide with flowering and early kernel filling stages of the grain (CAST, 2003). Because consumption of high levels of zearalenone can cause deleterious effects in domestic animals, ZEA and it's congeners are generally considered to be mycotoxins. ZEA is a resorcyclic acid lactone containing an unsaturated bond at C1'-C2' and a ketone function at position C6'. Either the double bond or the ketone, or both, can be reduced yielding a series of congeners and stereoisomers (Figure 1). The reductions are important because they affect the biological activity, as well as the physical properties of the molecule. Reduction is also important because it represents a mechanism whereby organisms can biotransform ZEA. As might be expected, not all of the products of biotransformation have equivalent physical characteristics or bioactivity. α-Zearalanol (α-ZAL), also known as zeranol (trade name Ralgro®) is used in some countries as a growth promoter in cattle due to it's anabolic activity. The oestrogenic activities of ZEA and related compounds will not be reviewed here, as they are the subject of a separate article in this issue. The potential effects of ZEA on human health have also been reviewed elsewhere (Altomare *et al.*, 2007; Massart *et al.*, 2010; Reddy *et al.*, 2010), as have field outbreaks and the implications for animal husbandry (EFSA, 2004; Kanora and Maes, 2009; Minervini and Dell'Aquila, 2008; Morgavi and Riley, 2007; Richard, 2007). Given the potential health effects of ZEA and related molecules in humans and domestic animals, food and feed have been, and continue to be,

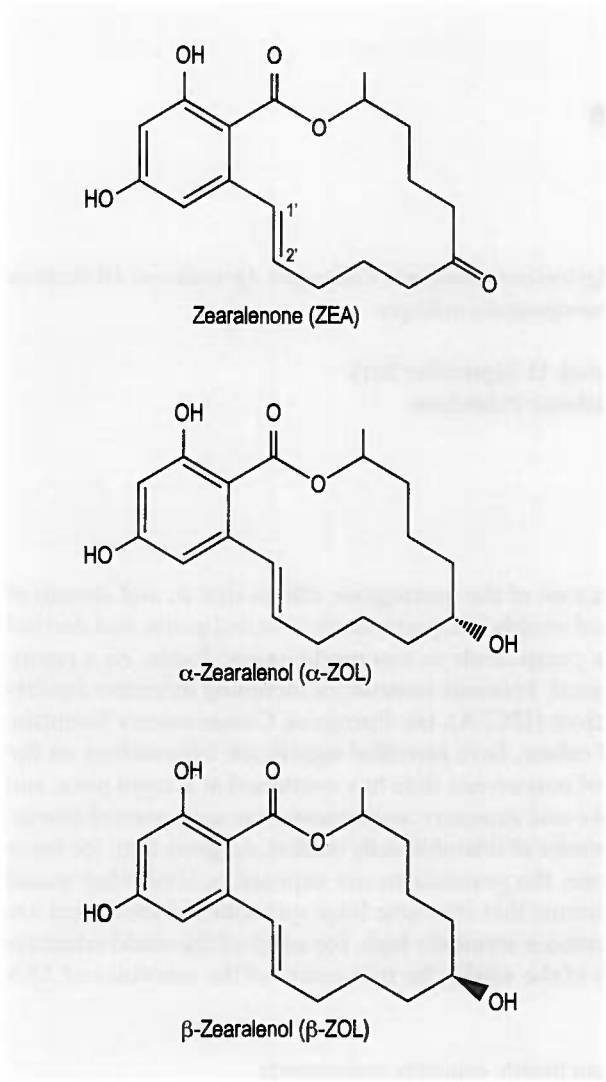

Figure 1. Schematic structures of zearalenone (ZEA), α-zearalenol (α-ZOL), and β-zearalenol (β-ZOL). Not shown are the corresponding congeners where the C1'-C2' double bond is reduced: zearalanone (ZAN), α-zearalanol (α-ZAL), and β-zearalanol (β-ZAL).

frequently tested for these compounds. Several nations have established guidance levels or maximum permissible levels for ZEA in foods, ranging from 50 to 1000 µg/kg (ppb) (FAO, 2004), and regulations relating to mycotoxins in food were recently reviewed (Van Egmond *et al.*, 2007).

The basis for assessing any type of exposure is detection and quantitation. The detection methods for ZEA are varied and encompass many techniques such as thin layer chromatography, liquid chromatography (LC), and gas chromatography in combination with various detectors. ZEA has a characteristic ultraviolet spectrum and is fluorescent, with the fluorescence intensity dependent upon the environment. Widely used methods for detecting ZEA include LC with ultraviolet (UV), photodiode array (PDA) or fluorescence (FL) detection, as well as mass spectrometry (MS) or tandem mass spectrometry (MS/MS). Increasingly LC-MS/MS is finding use for detecting ZEA along with other mycotoxins in multi-toxin screening assays. Other commonly used screening assays are those based upon antibodies (immunoassays), in particular enzyme-linked immunosorbent assays (ELISAs) or lateral flow 'dip stick' test strips. The performance characteristics of an assay (e.g. sensitivity, specificity, range, etc.) and whether it is a quantitative or qualitative test are important considerations when evaluating the results from the assay. In this review the data on ZEA occurrence have been collected by many laboratories using many different analytical protocols. While it is impossible in the scope of this review to fully characterise the performance characteristics of the method used in each report, interested readers are referred to the primary literature, to several excellent reviews of analytical methods for mycotoxins (Krska *et al.*, 2008; Shephard *et al.*, 2010) and to a recent book with chapters relevant to this subject (Rai and Varma, 2010).

Humans can be exposed to environmental oestrogens through a variety of routes. Most obvious is the direct exposure through consumption of contaminated foods or minimally processed commodities such as cereal grains. However, most food receives at least minimal processing, whether in the form of cooking or in combination with other ingredients to yield more complex foods. As such, this review includes a brief discussion of the effects of processing on ZEA. There are also less obvious routes by which humans may be exposed. These include through the environment (inhalation, or from drinking water) and secondary or indirect exposure through the consumption of the tissues (milk, meat) of domestic animals that have themselves been exposed to ZEA. Because of the many potential sources and routes of exposure, the exposure to ZEA and related metabolites is multifaceted.

Exposure to ZEA is not a new problem, and for this reason there have been periodic reviews and exposure assessments. Among major reviews of the various aspects of ZEA exposure and risk assessment are those that have been conducted by the Food and Agriculture Organization of the United Nations (FAO), in particular the Joint FAO/WHO Expert Committee on Food Additives (JECFA). The literature before 2000 on the prevalence and toxicology of ZEA was well reviewed in the JECFA report from that year (JECFA, 2000). Since that time there have been several other comprehensive reports summarising exposure (CAST, 2003; Reddy *et al.*, 2010; SCOOP, 2003; Yazar and Omurtag, 2008; Zinedine *et al.*, 2007). The SCOOP report (SCOOP, 2003) contains substantial occurrence data for participating countries from the European Union, as well as consumption data and estimates of dietary intake. Because of the wealth of data that has been summarised in other locations, and the large amount of data on ZEA occurrence that has been

published within the past few years this review will focus on literature published more recently than the JECFA and SCOOP reports or the review by Zinedine *et al.* (2007). Where data on occurrence has been found that was not included in one of these reports, it has been included here. For consistency with earlier reports the tables will follow the format of the JECFA report (2000).

2. Occurrence in human foods

The possible role of exposure to oestrogenic mycotoxins, including ZEA and α-ZAL, in the development of puberty has long been of interest, and was recently reviewed (Massart and Saggese, 2010; Sherif *et al.*, 2009). The need for further assessment of the health risks to children remains (Sherif *et al.*, 2009). Data on the prevalence of ZEA throughout the world are summarised in Table 1. The table presents much information, but caution should be exercised when contemplating data from the primary literature that has been distilled down to a minimum, as in this table. There are certain limitations of this form of presentation that bear discussion. First, given the global nature of the human food supply, the 'location' information is not as straight forward as it would appear. In many cases food that has been collected from a market in one location may have been produced quite a distance from the market. Therefore readers are cautioned not to assume that the location listed is the location from which the food originated, although in many cases that is no doubt true. The data set also tend to be biased towards countries or regions where substantial monitoring has been conducted. For example, there has been considerable monitoring in the member states of the European Union, but much less (published) monitoring in North America. Secondly, the incidence data reflect the number of 'positive' samples (D, or 'detected') divided by the total number of samples tested (T, or 'tested'). This is a useful statistic to provide an indication of prevalence, but is easily subject to manipulation. For example, by lowering the limit of detection (LOD) it is likely that more contaminated samples will be found and therefore the incidence will be higher. The LOD is, in turn, influenced by the technology used in the analysis and can vary dramatically. Likewise, the arithmetic mean of a data set is a simple statistic, but one that is not applied consistently by all authors. In some of the primary literature the mean level of contamination was reported as the mean of *all* of the samples, while in others the reported mean is the average of only the *contaminated* samples, and in certain cases which of these two methods of calculation have been used have not been specified. Likewise, reporting of the range is often not consistent. Some have reported the range as encompassing that between the lowest contaminated sample to the highest contaminated sample. Still others consider the minimum of the range to be the LOD or limit of quantitation (LOQ), and in many cases the full range has not been reported, only the value of the highest positive sample. Despite these issues the data set is very useful in giving a general indication of which commodities and foods represent the greatest potential sources for human exposure.

Grains

Maize (corn) clearly represents a large potential contributor to exposure, with a relatively high incidence of contamination. The mean levels of contamination in maize reported in Table 1 are clustered around concentrations between 10-100 µg/kg (13 studies), 200-500 µg/kg (8 studies), and below 6 µg/kg (9 studies). The maximum levels found in maize in these studies also tended to be rather high, many above 1000 µg/kg. The mean levels of contamination in wheat and barley (Table 1) are clustered around concentrations of 10-100 µg/kg (8 studies), and below 5 µg/kg (7 studies), with a few above 100 µg/kg (5 studies). The maximum levels found in wheat and barley in these studies also tended to be lower, with few above 1000 µg/kg. These factors tend to suggest that, in general, there is a lower level of ZEA contamination in wheat and barley than in maize. While Table 1 contains substantial information on the prevalence of ZEA in human foods and commodities, there are additional reports in the literature that, while not conducive to fitting into the format of Table 1, nevertheless reveal important aspects of ZEA exposure (Giraud *et al.*, 2010; Goliński *et al.*, 2010). A recent study was conducted of the susceptibility of winter wheat cultivars to *Fusarium* head blight in Poland. Two locations were compared with respect to the concentrations of mycotoxins, including ZEA, found in the kernels and chaff of inoculated wheat. Marked differences in ZEA concentrations were found at the two locations, and with the type of fraction, however generally the levels in the chaff were substantially higher than kernels classified as either healthy or *Fusarium* damaged (Goliński *et al.*, 2010). Oats and rye were commodities with relatively low levels of reported contamination. Of the 10 studies summarised in Table 1 for oats or rye, the sample with the greatest contamination contained 29 µg/kg ZEA. These data support the earlier observations that corn and wheat appear to be the commodities of greatest concern with respect to ZEA contamination (JECFA, 2000; Kuiper-Goodman, 1987). Fewer studies have been conducted with soybeans and rice, which makes generalisations more difficult. The studies of soybeans and soybean meal clearly indicate this as a potential source of exposure, the two studies in the Europe and Mediterranean region suggest modest levels of contamination, while the single study in Asia and Oceania had higher average contamination with ZEA.

Nuts, edible oils, miscellaneous foods and spices

Occurrence data in a variety of nuts was summarised in the JECFA document (JECFA, 2000), where few contaminated samples were reported. The exception was a report of contaminated oilseeds from Uruguay, of which 6 of 64

Table 1. Occurrence of zearalenone in commodities and human foods.

Food(s)	Location	Incidence D/T[a]	Mean[b] (µg/kg or µg/l)	Range	Reference
Grains					
Maize	Europe & Mediterranean	59/93	279	up to 1,958	Binder *et al.*, 2007
Maize	Bulgaria	4/19	80.6	up to 148	Manova and Mladenova, 2009
Maize	Croatia	41/49	3.84	0.43-39.12	Domijan *et al.*, 2005
Maize	Croatia	12/12	317	27.7-1,182	Šegvić Klarić *et al.*, 2009
Maize	Romania	38/70	NR[C]	up to 2,250	Macri *et al.*, 2009
Maize	Poland	8/42	391.5	30-1,344	Wiśniewska-Dmytrow *et al.*, 2004
Maize	France	23/56	26	3-165	Scudamore and Patel, 2009b
Maize	Italy	1995: 98[d]	1995: 79	up to 490	Pietri *et al.*, 2004
		1996: 104	1996: 453	up to 2,531	
		1997: 94	1997: 49	up to 590	
		1998: 114	1998: 13	up to 356	
		1999: 93	1999: 27	up to 280	
Maize	Spain	4/27	1.14	0.47-2.24	Jaimez *et al.*, 2004
Maize flour	Portugal	0/5	ND[e]	NA[f]	Cunha and Fernandes, 2010
Corn breakfast cereal	Portugal	1/4	49	NA	Cunha and Fernandes, 2010
Maize	Morocco	3/20	14	12-17	Zinedine *et al.*, 2006
Maize	Côte d'Ivoire	10/10	NR	20-50	Sangare-Tigori *et al.*, 2006
Maize flour	Iran	12/19	377	up to 889	Oveisi *et al.*, 2005
Maize snack	Iran	19/19	832	up to 1,471	Oveisi *et al.*, 2005
Maize	Nigeria	103/182 (ZEA)	49 (ZEA)	115-779 (ZEA)	Adejumo *et al.*, 2007
		23/182 (α-ZOL)	63.6 (α-ZOL)	32-181 (α-ZOL)	
Maize	Cameroon	31/40	69	28-273	Njobeh *et al.*, 2010
Corn flakes	Bahrain	3/5	3.1	NR	Musaiger *et al.*, 2008
Maize	Bangladesh	(10%)	1.5	up to 30	Dawlatana *et al.*, 2002
Maize	Korea	9/38	340	41-909	Thongrussamee *et al.*, 2008
Maize	Asia & Oceania	128/312	463	up to 6,468	Binder *et al.*, 2007
Maize	Argentina	21/58	NR	100-1,560	Roigé *et al.*, 2009
Maize	Argentina	13/26	15	3-42	Scudamore *et al.*, 2009b
Maize flour	Brazil	0/17	ND	NA	Sekiyama *et al.*, 2005
Maize grits	Brazil	0/7	ND	NA	Sekiyama *et al.*, 2005
Popcorn	Brazil	1/24	448	NA	Sekiyama *et al.*, 2005
Maize products	Brazil	0/74	ND	NA	Kawashima and Valente Soares, 2006
Maize	Mexico	17/24	NR	3-83	Briones-Reyes *et al.*, 2007
Maize breakfast cereal	Canada	7/34	6.1	up to 21	Roscoe *et al.*, 2008
Wheat	Europe & Mediterranean	44/48	187	up to 921	Binder *et al.*, 2007
Wheat	Croatia	4/6	29	13-50	Šegvić Klarić *et al.*, 2009
Wheat	Croatia	NR[g]	3.59 & 4.21	2.59-5.33	Ivić *et al.*, 2009
Wheat	Lithuania	32/100	NR	up to 95.6	Mankevičiene *et al.*, 2007
Wheat flour	Denmark	10/30	1	up to 2	Rasmussen *et al.*, 2003
Wheat	Belgium	NR/115	18.2	Up to 232	Harcz *et al.*, 2007
Wheat	Netherlands	NR/71	NR	up to 5,700	Hoogenboom *et al.*, 2008
Wheat	Poland	1/17	340	180-600	Wiśniewska-Dmytrow *et al.*, 2004
Wheat	Germany	11/181	69	20-250	Döll *et al.*, 2002
Wheat flour	Portugal	1/7	27	NA	Cunha and Fernandes, 2010
Wheat	Bulgaria	1/54	10	NA	Manova and Mladenova, 2009
Wheat	Ethiopia	0/16	ND	NA	Ayalew *et al.*, 2006
Wheat (crushed)	Bahrain	4/4	0.3	NR	Musaiger *et al.*, 2008
Wheat	Kenya-Nakuru	29/48	3.8	1.6-35	Muthomi *et al.*, 2008
Wheat	Kenya-Nyandarua	18/34	7.1	1-96	Muthomi *et al.*, 2008

Table 1. Continued.

Food(s)	Location	Incidence D/T[a]	Mean[b] (µg/kg or µg/l)	Range	Reference
Grains (continued)					
Wheat	Argentina	22/45	NR	NR	Roigé *et al.*, 2009
Wheat/bran	Asia & Oceania	26/98	165	up to 1,489	Binder *et al.*, 2007
Wheat breakfast cereals	Canada	11/29	2.4	up to 5.5	Roscoe *et al.*, 2008
Farro	Italy	0/8	ND	NA	Castoria *et al.*, 2005
Barley	Europe & Mediterranean	9/81	221	up to 970	Binder *et al.*, 2007
Barley	France	2/5	1.25 (median)	up to 3.4	Malmauret *et al.*, 2002
Barley	Poland	15/15	63	40-120	Perkowski *et al.*, 2003
Barley	Poland	0/12	ND	NA	Wiśniewska-Dmytrow *et al.*, 2004
Barley	Bulgaria	2/18	29	up to 36.6	Manova and Mladenova, 2009
Barley	Lithuania	38/66	(NR)	up to 193	Mankevičiene *et al.*, 2007
Barley	Croatia	4/4	62	35-84	Šegvić Klarić *et al.*, 2009
Barley	Ethiopia	0/15	ND	NA	Ayalew *et al.*, 2006
Barley	Tibet	24/25	161	25-270	Haubruge *et al.*, 2003
Oats	Europe & Mediterranean	0/11	ND	NA	Binder *et al.*, 2007
Oats	United Kingdom & Scandinavia	5/12	8	3-22	Scudamore *et al.*, 2007
Oats	United Kingdom	15/296	NR	up to 29	Edwards *et al.*, 2009
Oats	Croatia	1/2	18	18	Šegvić Klarić *et al.*, 2009
Oats	Lithuania	4/7	NR	up to 16.3	Mankevičiene *et al.*, 2007
Oats	Bahrain	1/5	0.3	0.3	Musaiger *et al.*, 2008
Oat breakfast cereal	Canada	3/27	4.7	6.9	Roscoe *et al.*, 2008
Rye	Lithuania	1/10	NR	up to 28.8	Mankevičiene *et al.*, 2007
Rye	Poland	0/22	ND	NA	Wiśniewska-Dmytrow *et al.*, 2004
Rye flour	Denmark	2/30	1	up to 2	Rasmussen *et al.*, 2003
Teff	Ethiopia	0/9	ND	NA	Ayalew *et al.*, 2006
Sorghum	Ethiopia	2/29	25	19-32	Ayalew *et al.*, 2006
Rice	Bahrain	8/10	0.7	NR	Musaiger *et al.*, 2008
Rice	Côte d'Ivoire	10/10	NR	50-200	Sangare-Tigori *et al.*, 2006
Rice/bran	Asia & Oceania	5/27	77	up to 162	Binder *et al.*, 2007
Rice breakfast cereal	Canada	2/29	2.9	up to 3.6	Roscoe *et al.*, 2008
Cereal grains	Finland	0/23	ND	NA	Eskola *et al.*, 2001
Cereal grains	Portugal	171/307	170	up to 930	Marques *et al.*, 2008
Multi grain breakfast cereal	Portugal	11/14	42	28-69	Cunha and Fernandes, 2010
Multi grain breakfast cereal	Canada	11/36	15	up to 100	Roscoe *et al.*, 2008
Legumes					
Soybean meal	Europe & Mediterranean	1/18	50	NA	Binder *et al.*, 2007
Soy foods	Germany	7/45 (ZEA) 5/45 (α-ZOL) 2/45 (β-ZOL)	47 (ZEA) 4.6 (α-ZOL) 5 (β-ZOL)	2-214 (ZEA) 2-11 (α-ZOL) 5 (β-ZOL)	Schollenberger *et al.*, 2007
Soybeans	Cameroon	0/5	ND	NA	Njobeh *et al.*, 2010
Soybean meal	Asia & Oceania	21/122	170	up to 1,078	Binder *et al.*, 2007
Peanut meal	Asia & Oceania	5/9	2,506	up to 4,587	Binder *et al.*, 2007
Peanuts	Cameroon	10/16	70	31-186	Njobeh *et al.*, 2010
Peanuts	Côte d'Ivoire	10/10	NR	50-200	Sangare-Tigori *et al.*, 2006
Edible oils					
Soybean oil	Germany	3/14 (ZEA) 1/14 (α-ZOL)	24 (ZEA) 8 (α-ZOL)	5-46 (ZEA) 8 (α-ZOL)	Schollenberger *et al.*, 2008

Table 1. Continued.

Food(s)	Location	Incidence D/T[a]	Mean[b] (µg/kg or µg/l)	Range	Reference
Edible oils (continued)					
Sunflower oil	Germany	0/16 (ZEA) 0/16 (α-ZOL)	ND ND	ND NA	Schollenberger *et al.*, 2008
Maize germ oil	Germany	9/17 (ZEA) 0/17 (α-ZOL)	505 (ZEA) ND	9-1,730 (ZEA) NA	Schollenberger *et al.*, 2008
Maize oil	Germany	4/6	99	57-135	Siegel *et al.*, 2010
Edible oils (non-maize)	Germany	0/63	ND	NA	Schollenberger *et al.*, 2008
Edible oils (non-maize)	Germany	0/40	ND	NA	Siegel *et al.*, 2010
Miscellaneous					
Figs (dried)	Turkey	7/52	NR	NR	Şenyuva and Gilbert, 2008
Alfalfa seeds	Lithuania	NR	25.8	NR	Kordusiene *et al.*, 2010
Flavor ingredients and spices	not specified	0/60	ND	NA	Boonzaaijer *et al.*, 2008
Paprika	Spain	25/64	NR	10-131	Santos *et al.*, 2010
Chilli	Spain	16/35	NR	10-129	Santos *et al.*, 2010
Beans	Cameroon	5/15	48	27-157	Njobeh *et al.*, 2010
Casava Flour	Portugal	1/1	14	NA	Cunha and Fernandes, 2010
Food supplements	Belgium	0/62	ND	NA	Di Mavungu *et al.*, 2009
Medicinal and aromatic herbs	Spain	82/84	NR	0.3-45	Santos *et al.*, 2009
Eggs	Belgium	7/20 (ZEN) 9/20 (β-ZOL) 11/20 (α-ZOL)	NR	All between the LOD & LOQ: 3-10 (ZEN) 6-20 (β-ZOL) 1.5-5 (α-ZOL)	Tangni *et al.*, 2009
Beef tissue (zearanol)	Japan	0/33	ND	NA	Furusawa and Kishida, 2006
Foods[h]	Switzerland	29/225	NR	2-22.6	Rhyn and Zoller, 2003
Foods[i]	Germany	7/15	16	4.9-45	Cramer *et al.*, 2007
Foods[j]	Jordan	0/108	ND	NA	Salem and Ahmad, 2010
Foods	Tunisia	15.0%	10.4	1.8-41.6	Ghali *et al.*, 2008
Misc. foods	Cameroon	1/6	67	NA	Njobeh *et al.*, 2010
Moldy foods	Austria	2/87 (ZEN) 1/87 (ZEN-4-glc)	NR	up to 640 (ZEN) up to 62,000 (ZEN-4-glc)	Sulyok *et al.*, 2010

[a] Number of samples above the detection limit (D) divided by the total number of samples reported (T).

[b] The arithmetic mean of results, which may have been calculated based on the total number of samples or only the positive samples (see text).

[c] The indicated value was not reported (NR) in the citation. This may be the result of a different statistic being used (median *versus*. mean for example), the presence of multiple data treatments, or the absence of information in the article to make the determination.

[d] The total number of samples (T) for each year. Over all the years the percentage of positive samples was 44%. A breakdown of the number of positive samples was not provided for each year except 1996 (91%).

[e] Not detected (ND).

[f] Not applicable (NA). For example if there was only one positive sample, or none, there is no range to report.

[g] This was a trial comparing the effectiveness of nine fungicides in protecting against *Fusarium* head blight of wheat. The mean level of ZEA was 3.59 µg/kg in treatments versus 4.21 µg/kg in the untreated controls.

[h] Foods tested included: wheat, wheat flour, wheat bran, spelt, rye, breads, cookies, pasta, rice, muesli, oat flakes, cereal bars, maize products, corn flakes, barley, millet flakes, buckwheat flour, wild rice, lentil, and powdered infant food.

[i] Foods included maize flakes, tortilla chips, bread, taco shells, crackers, oat flakes and noodles.

[j] Foods included cereals, nuts, green coffee, legumes, sunflower seeds, sesame seeds.

samples were reported to contain greater than 100 µg/kg. As shown in Table 1 there are several more recent studies with peanuts, peanut meal and chestnuts. The studies with peanuts and peanut meal would tend to suggest that further research is needed, as both studies reported more than half of the samples were positive, and the highest concentration found (in peanut meal) was substantial: 4,587 µg/kg (Binder *et al.*, 2007; Njobeh *et al.*, 2010). Testing of edible oils is a more recent phenomenon and the prevalence of ZEA in edible oils made from maize, sunflower seed, soybeans, or other sources is summarised in Table 1. While the data set is limited, the situation with the oils roughly mimics the situation in the materials from which they are derived. That is, oil derived from maize germ showed a greater prevalence and a higher average level of contamination, than oil derived from soybeans or sunflower seeds (Schollenberger *et al.*, 2008). Other edible oils tested by the same group were: olive, rapeseed, safflower, wheat germ, pumpkin kernel, peanut, walnut, grape kernel, sesame seed, linseed, and palm, all of which (n=63) did not contain detectable ZEA or α-zearalenol (α-ZOL). A novel method for extracting ZEA from edible oils (Siegel *et al.*, 2010) may be helpful for future testing, which seems warranted. Using a per capita consumption estimate of 56 g of vegetable oils per day, 2 g of which is from corn oil, the daily intake of ZEA was estimated as 1.6 µg from non-refined corn germ oil (Schollenberger *et al.*, 2008). A wide variety of spices have also been examined for contamination, generally with very low or no contamination (JECFA, 2000). No ZEA was reported in 60 samples of flavour ingredients and spices that were examined, including: lime oil, orange oil, melon extract, grapefruit oil, paprika extract, tangerine oil, olive oil, chilli pepper oil, peppermint oil, galangal root powder, gentian root powder, chilli pepper, black pepper, white pepper, garlic powder, paprika powder, coriander seed, dill herb, and onion (Boonzaaijer *et al.*, 2008). Recent data on the occurrence in paprika and chillies in Spain (Table 1) suggests that ZEA may be prevalent in these spices, but at modest levels, as the highest positive sample contained 131 µg/kg. The presence of mycotoxins in botanicals and dried fruits was recently reviewed (Trucksess and Scott, 2008). ZEA has been found in ginseng root, although a survey of occurrence was not conducted (Gray *et al.*, 2004). Recently 39 types of medicinal or aromatic herbs purchased in Spain were tested for multiple mycotoxins, including ZEA (Santos *et al.*, 2009). Of the 84 samples that were tested, 82 were contaminated above the limit of detection (0.14 µg/kg), 40 contained 0.3 to 5 µg/kg, 22 contained 5.1 to 10 µg/kg, and 20 contained more than 10.1 µg/kg. Samples of frangula bark (*Rhamnus frangula*) and olive leaves, both contained over 40 µg/kg and contamination with multiple mycotoxins was common (Santos *et al.*, 2009).

Effects of processing

Commodities may be subjected to a wide range of treatments before they are transformed into human food. These can range from very minimal (for example physical separation), to various physical, chemical, or microbiological treatments. ZEA is relatively heat stable and in the absence of reaction to form conjugates, ZEA cannot be expected to substantially degrade during moderate thermal processing. The effects of processing on ZEA were reviewed (Ryu *et al.*, 2002), which included an examination of chemical treatments (such as ammoniation), physical processing (thermal processing, sieving, milling, extrusion cooking), and biological processing (i.e. fermentation). Exposure of ZEA to UV light also results in its degradation (Murata *et al.*, 2008). Baking and roasting, can substantially decrease ZEA, although given the thermal stability of the molecule much often survives the treatment as well. Sieving or de-hulling of grain can reduce ZEA content, and ZEA tends to associate in the bran and germ fractions of dry milled maize or wheat. The level of ZEA was substantially higher in red kernels which had been isolated from *Fusarium culmorum* infected wheat than in unsorted kernels (Neuhof *et al.*, 2008). Early studies on the effects of milling were summarised in the JECFA report (JECFA, 2000), and more recently the effect of milling of maize in commercial mills in the UK was reported (Scudamore and Patel, 2009a). A comparison of using a traditional stone milling process to a modern roller milling process of wheat indicated that the stone milling process was more effective at reducing ZEA (Palpacelli *et al.*, 2007). During wet milling of corn the ZEA tends to concentrate in the fractions that are used for animal feed, with little in the starch. Dehulling during the processing of oats resulted in ZEA reduction (Scudamore *et al.*, 2007). ZEA can be reduced during the extrusion of maize, perhaps due to the combination of high temperature, high pressure and severe shear (Cetin and Bullerman, 2005; Ryu *et al.*, 1999), losses appear to be greater when the starting material contains lower levels of ZEA or extrusion is at higher moisture levels (Scudamore *et al.*, 2008a). However, extrusion of naturally contaminated wheat flour at 140 to 180 °C resulted in little change in ZEA (Scudamore *et al.*, 2008b). Fermentation has also been shown to reduce ZEA (Mokoena *et al.*, 2005; Ryu *et al.*, 2002).

Animal tissues, milk and eggs

There is a substantial literature over the past 5 years on the occurrence of ZEA in commodities, mixed feeds, fermented feeds (such as silages), and non-fermented feeds. It is beyond the scope of this manuscript to review that information, and interested readers are referred to several recent overviews of the subjects of occurrence in animal feeds (EFSA *et al.*, 2004), silages (Storm *et al.*, 2008), and distillers dried grains, which are often used in animal feed (Zhang *et al.*,

2009). Field outbreaks in domestic animals associated with mycotoxins are the result of overexposure and have also been recently reviewed (Morgavi and Riley, 2007; Richard, 2007; Goliński *et al.*, 2009). Levels in feeds are of direct relevance to the exposure of animals and are of indirect relevance to humans that may consume tissues or biological fluids, such as milk, derived from exposed animals. Thus risk assessments for animals (Mantovani *et al.*, 2009) also have a bearing on human exposures. A risk assessment of endocrine-active compounds in feeds, including ZEA, was performed (EFSA, 2004). The EFSA report summarised studies on the carry-over of ZEA and it's metabolites in edible tissues of several species, including chicken, turkey, duck, rabbit, pig, and cow. Essentially, while ZEA and it's congeners may be found in muscle of experimentally exposed animals, the levels are generally low. However, very high levels of exposure in the diet can lead to residues in significant levels, above 100 µg/kg, in certain edible tissues such as the liver and kidney (summarised in JECFA, 2000). Furthermore, significant levels of α- and β-ZOL can occur in the same tissues. Interspecies differences may also play a role. A recent study of pigs fed a diet containing deoxynivalenol and ZEA did not observe carryover of ZEA, α-ZOL, or β-ZOL into the serum. However, carryover was seen into the bile and liver of the animals, with carryover factors of the combination of ZEA, α-ZOL, and β-ZOL of 0.0094±0.0123 and 4.0±2.2 for liver and bile, respectively (Goyarts *et al.*, 2007). JECFA (2000) calculated a theoretical maximum daily intake of 1.6 µg (0.02 µg/kg bw/day) of α-ZAL derived from a maximum residue limit of 10 µg/kg in cattle liver and 2 µg/kg in cattle muscle. Analysis of diets may be a better basis for evaluating exposure of pigs to ZEA than analysis of blood or bile (Dänicke *et al.*, 2008). Transmission into eggs of ZEA, or it's metabolites, was not observed in two studies using laying hens (Dänicke *et al.*, 2002; Sypecka *et al.*, 2004).

The transmission of ZEA into milk of lactating sheep, cows, and pigs is low, with fairly substantial dosages of ZEA resulting in low µg/l concentrations in the milk, unless exposures of the animals were very high (EFSA, 2004; JECFA, 2000). Data for milk from the UK was summarised in the SCOOP report (2003), and indicated 3% of samples were positive for ZEA, with a highest value of 5.5 µg/kg. The dietary intake from milk was estimated to be between 2.0 and 4.8 ng/kg bw/day for adult consumers and up to 46.5 ng/kg bw/day for infants. An exposure assessment for ZEA in dairy milk carried over from feed was recently developed based upon a quantitative Monte Carlo model (Coffey *et al.*, 2009). The mean concentration of ZEA in dairy milk was estimated to be 0.39 µg/kg. The 5^{th} percentile and 95^{th} percentile were calculated as 0.0002 and 2.5570 µg/kg, respectively. The simulated daily exposure from milk was calculated as approximately 43.5 pg/kg bw for males and 46.8 pg/kg bw for females. The 95^{th} percentile was calculated as 6,553 pg/kg bw/day (males) or 6,895 pg/kg bw (females). The estimates suggest exposure through milk is likely, but that the daily intake from this source can be expected to be relatively low. The concern also exists that exposure from this source is additive to that from other sources, such as cereal grains.

Toxin conjugates

ZEA can undergo biotransformation into the analogs shown in Figure 1, but the products are not limited to those pictured. ZEA and its congeners can undergo further reactions to yield derivatives or conjugates that have not generally been detected along with the parent molecule(s). As such these are often referred to as 'masked', 'hidden', or 'conjugated' derivatives. When these are produced by biotransformation they may also be considered biomarkers of exposure. The conjugated forms are of interest in part because of the potential for the conjugates to be hydrolysed during digestion, possibly releasing bioactive products such as ZEA, or the reduced derivatives. The masked mycotoxins were recently categorised into four main sources depending upon where in the environment the conjugation happens; in fungi, in the host plants, during food processing, or through biotransformation in animals (Berthiller *et al.*, 2009b). With regards to ZEA, the formation of the 4-sulfate can occur in fungal culture and in plants. Glucosylation can also be mediated by fungi and plants, yielding glucopyranosides and diglucosides. The variety of potential products is substantial and has been well summarised by Berthiller *et al.* (2006, 2009a,b). In mammals, the glucuronide conjugate can also be formed, as can the sulfate, making these potential biomarkers of exposure. The use of enzymes in the synthesis of ZEA glucuronides was recently described (Stevenson *et al.*, 2008). The conjugates of ZEA are of significant interest both as possible sources of (indirect) exposure and as biomarkers of exposure to ZEA. The extent of their occurrence and contribution that they might make to the toxicity of contaminated foods require further investigation.

Other sources: beverages, airborne exposure, soil

Added to exposures from food sources are those from consumption of beverages and other, non-food sources. In addition to milk, other beverages that have been reported to be contaminated with ZEA are water, and beer. ZEA may occur in water through run-off from contaminated fields and has been detected in a variety of waters including drainage water, river water, at wastewater treatment plants, and in groundwater. Countries where it has been detected in water include Italy, Switzerland, Poland, and Portugal (Gromadzka *et al.*, 2009; Hartmann *et al.*, 2007, 2008; Laganà *et al.*, 2004; Russell and Paterson, 2007). In these studies the maximum levels ranged from 15 ng/l to 43.7 ng/l, indicating that the level of contamination is quite low. Interested readers are directed in particular to the report by Hartmann *et al.* (2008), where the levels of ZEA

in contaminated wheat and maize fields were determined, as were the levels in soil and in water draining from the fields. The level of contamination depended on the crop and the assessment period, and ranged from 0.1 to 4.3 mg/ha. This corresponded to between 0.001 and 0.070% of the amount calculated to be present in the plants (Hartmann *et al.*, 2008). An excellent review of ZEA in environmental samples, including water and soil, was recently published (Hoerger *et al.*, 2009). Because of the low levels that have been found, the contribution of ZEA from drinking water is expected to be low. The significance may be to aquatic or soil-dwelling animals that may have additional routes of exposure (Chen *et al.*, 2010).

In addition to water, humans have developed an affinity for beverages derived from fermented grains. As discussed above, levels of ZEA may be affected by fermentation and it is a natural question as to the extent to which ZEA is carried over into beer. Early literature on ZEA in beers was summarised in the JECFA report (JECFA, 2000). Carry-over from contaminated guinea-corn into a native Nigerian beer (*burukuru*) was reported to be 51% (Okoye, 1987). Intake in the UK from beer was estimated to be up to 0.3 ng/kg bw/day among consumers (SCOOP, 2003). A survey of two types of beer in Kenya (Pilsner, Tusker) indicated that all of 75 samples tested contained ZEA, with mean levels of contamination of 7.84 ng/l (Tusker) and 8.5 ng/l (Pilsner). The concentrations found ranged from 4.3 to 10.2 ng/l (Mbugua and Gathumbi, 2004). The fate of ZEA during the malting and brewing of barley-based beer was recently reported (Lancova *et al.*, 2008). With barley artificially inoculated with *Fusarium* spp. the ZEA level, initially 77 µg/kg in the malt grist, was reduced to 7 µg/l in the beer. When malt grist that contained less than 5 µg/kg of ZEA were used, the resulting beer contained less than 5 µg/l. In the SCOOP report (SCOOP, 2003), the UK was the only country to report data in beer, with dietary intake estimated to be up to 0.3 ng/kg bw/day among consumers. The data, taken together, suggest that in most cases exposure to ZEA through beer can be expected to be low although it should not be discounted as a minimally processed beer produced from highly contaminated grain might attain significant levels.

While ZEA has been rarely found as a contaminant of indoor air (Jarvis and Miller, 2005), the occurrence of ZEA in crop residues and soil likely contribute to the presence of ZEA in dusty environments. Therefore occupational exposures for those working in agricultural environments need to be considered. Dust collected from grain/corn storage facilities was found to be frequently contaminated with ZEA, with an average concentration of 126 µg/kg (Mayer *et al.*, 2007). In that same report the airborne concentration was 1 ng/m^3. Grain dust was also collected from farms and storage companies in Belgium (Tangni and Pussemier, 2007). Nine of 14 dust samples were above 0.05 µg/kg of ZEA and all were above 0.175 µg/kg of zearalenol. The median levels were 0.2 µg/kg (ZEA) and 1.1 µg/kg (ZOL), while the maximum levels were 2.4 µg/kg (ZEA) and 3.3 µg/kg (ZOL). The median intake of ZEA by inhalation was estimated to be 0.1% of the tolerable daily intake of 200 ng/kg bw/day. ZEA was also found in samples of air collected from poultry houses (Wang *et al.*, 2008). Based upon the levels found in the dusts, inhalation exposures for workers in the poultry house were calculated as 17.4 to 20.5 ng/day, while exposures for chickens were calculated as 0.436 to 0.513 ng/day.

3. Exposure and risk assessments

Estimates of ZEA intake and exposure have been developed and continue to be developed as technologies to detect ZEA expand, allowing for increased monitoring. An early assessment in Canada estimated dietary intakes of 0.19 µg/day (12-19 year old males) and 0.47 µg/day (1-4 year old children) (Kuiper-Goodman *et al.*, 1987). Several years later the mean intake for Canadian adults was estimated as <0.98 µg/day for 60 kg adults (<0.016 µg/kg bw/day, JECFA, 2000). For infants aged 6-9 months the estimated mean intake was <0.52 µg (<0.06 µg/kg bw/day). The JECFA report (2000) summarised estimated intakes reported by Eriksen and Alexander (1998) for Denmark (0.48 µg/day), Sweden (1.2 µg/day), Finland (1.3 µg/day), and Norway (1.5 µg/day). Furthermore, the authors of the JECFA report developed estimates for two populations within the United States of America: all of those aged 2 or older (<1.7 µg/day), and 20 to 39 year old men (<2.1 µg/day). The SCOOP report (SCOOP, 2003) contains a wealth of information on data from nine European countries. The estimates were divided into two categories based on occurrence data from either all samples (mean1) or occurrence data of only positive samples (mean2). The estimates were further subdivided by country and age of the population group. For intake estimates of specific countries and age groups, interested readers are referred to the SCOOP report itself. Using occurrence data based on mean1 (average of all samples), the mean intakes for adults ranged from 0.8 ng/kg bw/day (Italy) to 29 ng/kg bw/day (French males). For children the mean intakes ranged from 6.5 ng/kg bw/day (Germany, infants) to 54.8 ng/kg bw/day (ages 4-6 in the UK). A worst case scenario of Norwegian infants consuming porridge of one type, contaminated with ZEA, gave an intake estimate of 1,508 ng/kg bw/day: well above the estimates for most other population groups. Using the occurrence data based on mean2 (average of positive samples) the intake estimates ranged from 1.9 ng/kg bw/day (Italian adults) to 116.3 ng/kg bw/day (Austrian adults), with most estimates below 100 ng/kg bw/day. An important point, made in the SCOOP report, is that basing the risk assessment only on the average intakes would lead to an underestimation of the risk, as it neglects sub-populations that consume high amounts of the foods that might represent the greatest sources of contamination.

Additional data from Europe came with intake estimates developed from the French total diet study (FTDS; Leblanc *et al.*, 2005). Food samples (2,280) were used to make 456 composite samples. Samples were prepared 'as consumed' before compositing and analysis. Of the composite samples 245 were tested for ZEA, and only 5 of these exceeded the detection limit. Mean exposure for adults (15 years and older) was estimated as 33 ng/kg bw/day, while for children (3-14 years), it was estimated as 66 ng/kg bw/day. For adults the greatest exposure was estimated to be from bread (28.7% of the total), while for children it was estimated to be from breakfast cereals (23.1% of the total). For vegetarians the estimated average ZEA intake was between 50-200 ng/kg bw/day (Leblanc *et al.*, 2005). For Switzerland a total of 225 samples of foods (flour, corn, bread, pasta) were examined, and 13% were found to contain ZEA (range 2-22.6 µg/kg). The mean intake for the Swiss population was estimated to be <1 µg per capita/day (<0.02 µg/kg bw/day) (Rhyn and Zoller, 2003). Recently a comparison was made between daily intakes for consumers of organic versus conventional foodstuffs in Belgium (Harcz *et al.*, 2007). The foods were wheat-based, and the assumption was made that contaminant levels did not change during processing or food preparation. For ZEA the estimated intake from organic foodstuffs was 0.03 µg/day compared to 0.06 µg/day for consumers of conventional foodstuffs.

The dietary intakes of males, females, and young men in New Zealand to a range of xenoestrogens in food were estimated by Thomson *et al.* (2003). For ZEA the estimated exposures were 0.97 µg/day, 0.75 µg/day, or 1.2 µg/day for males (25 years and older), females (25 years and older), and young males (19-24 years), respectively. Xenoestrogenicity from dietary intake was almost equally attributed to natural and synthetic xenoestrogens. One approach to estimating health impacts involves an integrated probabilistic risk assessment (IPRA) model. Recently an IPRA model was used to estimate the health impacts on humans caused by crops contaminated with ZEA (Muri *et al.*, 2009). The model integrated the distribution characterising toxin exposure with a distribution characterising the susceptibility of individuals to toxic effects, and used occurrence data from Denmark and the Czech Republic. The outcome of the model was an individual margin of exposure, which, in the scenarios that were examined, indicated ZEA did not have an impact on human health (Muri *et al.*, 2009).

This conclusion, is of course, limited to the population that was studied, and in other populations the results might be different. In a study of occurrence in 10 samples each of maize, rice, and peanuts from Côte d'Ivoire (Ivory Coast) the occurrence and intake of ZEA from these sources was estimated (Sangare-Tigori *et al.*, 2006). All of the 30 samples were positive for ZEA, with levels up to 200 µg/kg (Table 1). Based on a weekly consumption of 500 g maize, 3 kg rice, and 150 g peanuts, the ZEA weekly intake was estimated as 655 µg, or 1.56 µg/kg bw/day assuming a body weight of 60 kg. This level of exposure (93.5 µg/day) is substantially higher than in the various estimates for Europe, Canada, USA and New Zealand which, as noted above, generally were calculated to be below 2 µg/day.

In order to help gauge the relevance of ZEA to human health, the estimated intakes can be compared to the intakes at which toxic effects might be expected to be seen. Or, more commonly, to levels that are deemed tolerable. Such levels have been derived from toxicity tests in animals and are often adjusted with 'safety factors'. Safety factors are commonly used in order to accommodate what are likely to be large difference in susceptibility of the population to the toxic effects, and to provide a margin for the extrapolation from toxicity studies in experimental animals to humans. The estimate of a tolerable intake can also depend upon the toxicological endpoint selected (oestrogenic effect, tumorigenicity, etc.). Not surprisingly, the 'safe levels' that result can be highly variable and are represented by many different statistics. In the case of ZEA, several estimates of safe or tolerable intakes have been developed. An early estimate of a tentative tolerable daily intake (t-TDI), based upon the non-hormonal effect level for ZEA, was 0.10 µg/kg bw/day, while the virtual safe dose, based upon tumorigenicity data was calculated as 0.05 µg/kg bw/day (Kuiper-Goodman *et al.*, 1987). Several years later, two additional estimates were published. One of these, by the Scientific Committee on Food, established a t-TDI of 0.2 µg/kg bw/day (SCF, 2000). A second, developed by JECFA established a provisional maximum tolerable daily intake (PMTDI) for ZEA of 0.5 µg/kg bw/day (JECFA, 2000). Both the PMTDI established by JECFA and the t-TDI value established by the Scientific Committee on Food were based upon the no-observed-effect-level (NOEL) of 40 µg/kg bw/day obtained from a 15 day study of oestrogenic effects in pigs. Thus the estimates of safe exposure cluster around the range of 0.05 to 0.5 µg/kg bw/day. For adults, assuming a 60 kg body weight, this equates to 3 to 30 µg/day. The intake estimates, summarised above, suggest most of the populations for which estimates have been made are, on average, below 2 µg/day. This would suggest that, on average, these adult populations are being protected from exposures to ZEA that might cause adverse effects. The issues arise with the fact that no one is average. Those who consume the types of foods that are more susceptible to contamination will likely have higher exposures, and those foods may occasionally have concentrations of ZEA that substantially exceed the average. Furthermore children, who may have a less varied diet than adults may be exposed to relatively larger amounts of ZEA (as noted with the intake estimates above). This, combined with a different level of susceptibility due to their stage of growth, suggests that children are a more susceptible population to ZEA.

4. Conclusions

The occurrence of ZEA and its metabolites varies widely between foods and, within a food type, between geographical locations. As seen with previous reports, cereal grains represent major sources of intake, although other sources such as milk or tissues (liver) cannot be discounted. Various regions of the world have different consumption patterns for foods, for example in certain regions consumption of wheat exceeds that of maize, while in other regions the situation is reversed. Thus, intake estimates are most relevant to local populations, and extrapolation to other populations is likely to bias results. This is relevant, because by far the greatest amount of data on occurrence (and therefore exposure) originates from the European Union and Canada. For those regions average intake estimates suggest exposures are generally below the levels deemed 'tolerable'. This is not to imply that those regions may not have problems with ZEA, particularly in sub-populations that consume large amounts of the foods that are most susceptible to contamination or in years where ZEA contamination is extraordinary. Furthermore while this analysis has dealt solely with ZEA and its metabolites, synergistic effects among mycotoxins and between mycotoxins and other xenobiotics could be important. The situation for many other parts of the world, for which there is much less data on occurrence, and therefore fewer estimates of exposure, is much more poorly defined. Because of this, exposure estimates remain an important research need around the world, as is the development of tools to help assess the impact of potential additive or synergistic effects.

Disclaimer

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